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Elevated Pulmonary Artery Systolic Pressures are Associated with a Lower Risk of Atrial Fibrillation Following Lung Transplantation

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Abstract

Background—Atrial fibrillation (AF) is common after open-chest procedures, but the etiology remains poorly understood. Lung transplant procedures allow for the study of novel contributing factors.

Methods—Records of lung transplant procedures performed at a single center between 2002 and 2009 were reviewed.

Results—Of 174 patients, 27 (16%) had AF a median 6 days post-surgery. Post-operative AF patients less often had right ventricular hypertrophy (RVH) by either electrocardiogram (0 versus 14%, p=0.042) or echocardiography (19% versus 47%, p=0.006), and had lower pulmonary artery systolic pressures (PASP) (39 ± 12 versus 51 ± 22, p=0.005). After multivariable adjustment, every 10 mmHg increase in PASP was associated with a 31% reduction in the odds of post-operative AF (OR 0.69, 95% CI 0.49-0.98, p=0.035). A higher pulmonary pressure was the only predictor independently associated with less post-operative AF.

Conclusions—Higher PASP was associated with a lower risk of AF after lung transplantation.

Keywords

Pulmonary artery systolic pressure; atrial fibrillation; lung transplantation; post-operative; right ventricular hypertrophy

Introduction

Atrial fibrillation (AF) is common after thoracic surgery, leading to an increase in health care resource utilization.[1-4] The etiology is thought to be related to inflammation,[5-8] and possibly an increase in sympathetic drive,[9] but hemodynamic alterations may also be important.[10] Post-operative AF has been reported to occur in 18-39% of lung transplantation procedures, and a few studies have described risk factors and possible mechanisms in this

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population.[11-15] Lung transplant recipients are a unique population with respect to manipulation of the left atrium and cardio-pulmonary hemodynamics. Understanding the predictors of post-operative AF in this specialized population may reveal mechanisms important to all AF in general. By analyzing the recent University of California, San Francisco (UCSF) experience with post-operative AF in a large number of pulmonary transplant patients over a relatively short period of time, we sought to further advance the understanding of post-operative AF in the lung transplant setting.

Methods

Data were collected from electronic and paper charts for patients who underwent lung transplantation at UCSF from 2002 through 2009 (n=184). Ten patients with pre-operative AF were excluded from the analysis. Of these 174, six were heart and lung transplants. Demographic data included age, sex and race. Past medical history was noted for the presence of diabetes, hypertension, congestive heart failure, and coronary artery disease. With respect to indication for transplantation, interstitial lung disease was defined on a clinical and not a pathologic basis.

The most recent elecrocardiograms (ECGs) and echocardiographic reports were used. Echocardiographic data collected included left ventricular ejection fraction, right and left ventricular and left atrial sizes, pulmonary artery systolic pressure and ventricular hypertrophy. Echocardiographic left atrial volumes was determined by Simpson's rule per the American Society of Echocardiography recommendations (available in 122, 88% of echocardiograms with atrial measurements), and designation of left atrial size was determined by left atrial volume indexed to body surface area: small <16 ml/m2; normal 16-28 ml/m2; mildly enlarged 29-33 ml/m2; moderately enlarged 34-39 ml/m2; and severely enlarged >39 ml/m2 [16]. For 16 patients (12%), echocardiograms did not include atrial volume measurements; in those cases, left atrial size was determined by a qualitative assessment by a physician board certified in both cardiovascular disease and echocardiography.

Echocardiographic evidence of left ventricular hypertrophy was defined as present if the left ventricular mass determined by area-length formula indexed by body surface area was mild or greater, corresponding to =103 gm/m^2 in men and =89 gm/m^2 in women.[16] Right ventricular hypertrophy was defined by a right free wall thickness of at least 0.5 cm in the left parasternal long axis view or subcostal view.[17]

For all echocardiograms, pulmonary artery systolic pressure was estimated by adding the right atrial pressure (determined by assessment of the inferior vena cava) to the systolic gradient between the right ventricle and right atrium determined by the maximum velocity of the tricuspid regurgitation jet via the Bernoulli equation.[18]

Peri-operative statins, beta blockers, ACE inhibitors, steroids, amiodarone and vasopressors received within seven days prior to or after undergoing transplantation were recorded. Post-operative complications including the occurrence of hypotension requiring intervention, infection, and clinical pericarditis were recorded.

Institutional review board approval was obtained for the study by the UCSF Committee on Human Research.

Statistical Analysis

Normally distributed continuous variables are presented as means ± SD; continuous variables that were not normally distributed are presented as medians and interquartile

ranges (IQR). Continuous variables were compared using t-tests and Wilcoxon Rank Sum tests as appropriate. Categorical variables were compared using the $?^2$ test. Multivariable analysis was performed using logistic regression analysis, with the output presented as odds ratios (OR) and 95% confidence intervals (95% CI). Covariates were selected for inclusion in the multivariate model based on convention or "face value" (e.g., age, sex, and indication for transplant) or if they were associated with both the predictor and outcome with a p value of <0.1. Two-tailed p values < 0.05 were considered statistically significant. Statistical analysis was performed using Stata version 9.2 (College Station, TX).

Results

Twenty-seven patients (16%) developed post-operative AF a median 6 days after their transplant surgery (IQR 3-7 days, total range 2-14 days). Table 1 shows the patient characteristics of those with and without post operative AF. Post-operative AF patients were significantly older and nearly significantly more often male. No other differences in demographics or past medical history were found to be statistically significant, including race (when dichotomized into White versus non-White, p value=0.09). The median duration of AF was less than one day (IQR 0-1 day), and the longest episode was 5 days. Seven patients underwent cardioversion, and the remainder self-terminated. All those that self-terminated did so within 2 days.

ECG and echocardiographic data in those with and without post-operative AF are shown in Table 2. The ECGs were performed a median 1 day (IQR 0-4 days) before the transplant procedure, and the echocardiograms were performed a median 1 day (IQR 0-7 days) prior to the procedure. None of those noted to have a small left atrium on echocardiography developed post-operative AF, but potentially due to the relatively small numbers of those with a small left atrium, this did not reach statistical significance (when left atrial size was dichotomized into small versus not small, the p value=0.10, and when dichotomized into small/normal versus enlarged, the p value=0.08). When restricting the analysis to the 122 participants with quantitative left atrial measurements, those with post-operative AF had a larger baseline left atrial volume (42 ± 14 ml) than those without post-operative AF (34 ± 15 ml), p=0.049. Right ventricular hypertrophy (RVH) by both ECG and echocardiography were less common in patients with post-operative AF. In addition, patients with post-operative AF exhibited significantly lower pulmonary artery pressures (PASP). Of note, a small left atrium was associated with significantly higher PASP: 66 ± 30 mmHg in those with a small left atrium versus 47 ± 20 mmHg, p=0.001. Among those with left atrial volume measurements available, every 10 mmHg increase in PASP was associated with a significant 1.6 ml smaller left atrial volume (95% CI 0.4 to 2.8 ml smaller left atrial volume, p=0.011).

Pre-operative receipt of statins, ACE inhibitors, steroids, beta-blockers and amiodarone did not differ significantly between those that did and did not develop post-operative AF. With respect to the same medications used within the 7 days after surgery, only amiodarone was more common in the post-operative AF group (24 out of 27 [88.9%] patients with post-operative AF versus 15 out of 146 [10.3%] patients without post-operative AF; p<0.001) — further examination revealed that this was due a greater number of AF patients being treated with amiodarone subsequent to AF onset.

The indication for transplant in those with and without post-operative AF is shown in Table 3. No significant differences were observed (overall ?2 p value=0.46). Although a larger proportion of those receiving single lung transplants developed post-operative AF (7 [24%]), this was not statistically significantly greater than those receiving dual lung transplants (20 [16%], p=0.16). Those with post-operative AF were not more likely to have post-operative

infection, hypotension requiring intervention, or clinically evident pericarditis, and did not exhibit any significant differences in hospital length of stay.

After adjusting for potential confounders, including age, sex, and indication for transplant, RVH was associated with a statistically significant 64% reduced odds of post-operative AF (or 0.36, 95% CI 0.13-0.99, p=0.049). A sensitivity analysis excluding the 6 patients with both heart and lung transplantation did not significantly alter this association. Consistent with the known biological relationship, RVH was collinear with pulmonary artery systolic pressure. An elevated PASP remained significantly associated with a lower odds of post-operative AF after adjustment for age, sex, and indication for transplant: every 10 mmHg increase in PASP was associated with an average 31% reduction in the odds of developing post-operative AF (OR 0.69, 95% CI 0.49-0.98, p=0.035). Again, a sensitivity analysis excluding the 6 patients with both heart and lung transplantation did not significantly alter this association. Whether assessing RVH as the primary predictor or PASP as the primary predictor, the association between age and post-operative AF was lost when age, sex, and indication for transplant were included in the multivariate model, making evidence of pulmonary hypertension the only predictor that was independently associated with post-operative AF (in an inverse relationship). When left atrial size was added to the model as a potential mediator, the statistical significance of the relationship between PASP and post-operative AF was attenuated (p=0.088), but the odds ratio did not meaningfully change: the OR went from 0.69 (95% CI 0.49-0.98) to 0.68 (0.44-1.06); this is more consistent with a loss of precision of the estimate (consistent with the fact that detailed left atrial size measurements were only available in 138 patients, 20 with post-operative AF) rather than strong statistical evidence that a small left atrial size mediates the association between higher pulmonary pressures and a lower risk of post-operative AF. However, when restricting the mediation analysis to the 122 participants with available left atrial volume measurements, addition of left atrial volume as a continuous variable to the multivariate model attenuated the association between elevated pulmonary artery pressures and post-operative AF, consistent with a mediation effect of left atrial size on the causal pathway between elevated pulmonary pressures and risk of AF: the OR for post-operative AF among these patients increased from 0.68 (95% CI 0.46-1.00) to 1.01 (95% CI 0.98-1.04).

Discussion

In our series of 174 patients without preexisting AF undergoing pulmonary transplant procedures, 27 (16%) developed post-operative AF. An elevated PASP was associated with a lower risk of post-operative AF after lung transplantation before and after adjusting for potential confounders. Corroborating the PASP data was the finding that echocardiographic evidence of right ventricular hypertrophy was also independently associated with a lower risk of post-operative AF in these patients. Evidence of higher pulmonary pressures was the only predictor independently associated with the risk of post-operative AF.

Post-operative AF occurs after open heart surgery in up to 37% of patients[1, 2, 19] and after lung transplantation in 18-39% of patients.[11-15] Post-operative AF is associated with prolongation of hospital stay, increased general health care resource utilization, and an increased risk of systemic thromboembolism and hemodynamic instability.[1, 2, 20] The exact mechanism of post-operative AF remains unknown, but left atrial enlargement is known to be associated with a higher risk for non-surgical AF,[21] and an enlarged left atrium may be an important risk factor for post-operative AF.[22] However, to our knowledge, a small left atrium has never been shown to be protective against post-operative AF.

To our knowledge, only one previous study has demonstrated an association between higher pulmonary pressures and less AF: Srimachahota et al. reported that an elevated PASP was

associated with an absence of AF in patients who had mitral commisurotomy for mitral valve stenosis.[10] As this previous report was a cross-sectional study and can therefore not distinguish cause and effect, ours is the first report to show that elevated pulmonary pressures negatively predict incident AF.

The reason that an elevated pulmonary pressure might protect against post-operative AF in these patients remains unclear. One potential mechanism may be that elevated right-sided pressures prevent expansion of the left atrium. Those with a small left atrium did exhibit significantly higher PASP, and none of the patients with a small left atrium developed post-operative AF. When atrial size as an ordinal variable (small, normal, mildly enlarged, moderately enlarged, and severely enlarged) was added to the multivariate model, the statistical significance of the association between PASP and post-operative AF was lost, but the odds ratio did not substantially change—this therefore may have been due to a loss of power rather than clear evidence of an intermediary effect. However, when the analysis was restricted to those with left atrial volume measurements, a substantial mediation effect was observed (ie, consistent with the notion that higher pulmonary pressures lead to a lower risk of post-operative AF via a smaller left atrium). This mediation effect supports the conclusion that high pulmonary artery pressures predict a lower risk of AF by providing evidence of a biologically plausible mechanism. It is also possible that the mechanism may be unrelated to effects on atrial size. For example, perhaps the hemodynamic benefit of lung transplantation is greater for those with higher pulmonary pressures, leading to a lower risk of post-operative AF.

The finding that elevated pulmonary pressures predict a lower risk of post-operative AF is potentially clinically relevant for several reasons. First, as this appears to be mediated by a small left atrium and as these patients essentially by definition have elevated right atrial pressures, this would provide compelling evidence that the left atrium (and specifically left atrial size) confers AF risk whereas the right atrium does not. Why left (and not right) atrial enlargement would be particularly important remains unknown. However, left atrial and/ or pulmonary vein stretch may be important to pulmonary vein triggering,[23-25] a phenomenon known to be important in spontaneous AF,[26] which may be prevented in the setting of pulmonary hypertension. It is also important to emphasize that, while this data supports a stronger left atrial effect on AF risk in this particular setting, it by no means proves that left atrial processes alone are both necessary and sufficient for AF in general; for example, previous evidence has demonstrated that the right atrium may drive AF in some cases.[27, 28]

Mason et al. observed a higher rate of AF in their patients with primary pulmonary hypertension, a finding that might appear to be contradictory to ours.[13] However, they found an association among patients with a diagnosis of primary pulmonary hypertension and not higher pulmonary pressures per se. Of note, none of our primary pulmonary hypertension patients developed post-operative AF (as shown in Table 3), and therefore some unmeasured differences may be present. Importantly, they also included patients with both AF and atrial flutter (the former known to be a rhythm involving the right atrium in the majority of cases), whereas we focused only on AF.

Our study has several limitations. First, because the study was retrospective, our measurements (such as echocardiographic measurements) were not performed per a prospectively determined protocol. PASP was derived from echocardiography. Although this has been validated and is considered accurate,[18, 29, 30] we did not use the reference standard, right heart catheterization. Adequate left atrial measurements were not available in all patients, potentially limiting our ability to assess left atrial size as a mediator of the PASP-AF association. In addition, the heterogeneous nature of some of these measurements (some atrial sizes were determined quantitatively and some determine qualitatively) may

have reduced the accuracy and precision of our results. While we used previously established methods to determine echocardiographic evidence of RVH,[17] these have not been as rigorously studied as other echocardiographic measurements; however, we believe the consistent findings between RVH detected by either ECG or echocardiogram and post-operative AF as well as the separate finding linking elevated pulmonary pressures and AF provide some internal validity. Finally, we cannot exclude the possibility that insufficient power due to the relatively small absolute number of patients with AF was responsible for some of our negative findings (such as the fact that age was no longer a statistically significant predictor of post-operative AF after multivariable adjustment).

In conclusion, our review of lung transplant recipients shows that post-operative AF occurred in 16% in the immediate post-operative period. A higher PASP was associated with a lower risk of post-operative AF.

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Table 1

Demographic characteristics of patients with and without post-operative atrial fibrillation.

Characteristic	No post-opertaive atrial fibrillation (n=147)	Post-operative atrial fibrillation (n=27)	p value
Age (mean years +/- SD)	48.5 +/- 13.04	55.5 +/- 10.21	0.009
Male	80 (54.4 %)	20 (74.1 %)	0.058
Race			0.36
White	108 (73.5 %)	24 (88.9 %)	
Black	7 (4.8 %)	1 (3.7 %)	
Hispanic	13 (8.8 %)	0 (0 %)	
Asian	6 (4.1 %)	0 (0 %)	
Other	13 (8.8 %)	2 (7.4 %)	
Hypertension	35 (23.8 %)	8 (29.63 %)	0.52
Diabetes	16 (10.88 %)	5 (18.52 %)	0.26
Coronary artery disease	10 (6.85 %)	2 (7.41 %)	0.92
Congestive heart failure	9 (6.12 %)	1 (3.7 %)	0.62

Table 2

Echocardiographic and electrocardiographic findings in patients with and without post operative atrial fibrillation.

Electrocardiographic Characteristic	**No post-operative atrial fibrillation (n= 147)**	**Post-operative atrial fibrillation (n= 27)**	**p value**
Right atrial enlargement	22 (14.97 %)	3 (11.11 %)	0.60
Left atrial enlargement	17 (11.56 %)	2 (7.41 %)	0.52
Right ventricular hypertrophy	20 (13.61 %)	0 (0 %)	0.042
Left ventricular hypertrophy	7 (5 %)	1 (4 %)	0.81
Echocardiographic Characteristic			
Left atrial size[a]			0.17
Small	13 (11%)	0	
Normal	96 (81%)	16 (80%)	
Mildy enlarged	7 (6%)	3 (15%)	
Moderately enlarged	2 (2%)	1 (5%)	
Left ventricular hypertrophy	7 (5%)	1 (4%)	0.81
Right ventricular hypertrophy	69 (46.94 %)	5 (18.52 %)	0.006
Pulmonary artery systolic pressure (mean mmHg ± SD)	51 ± 22	39 ± 12	0.005

[a]Available for 138 patients (20 with post-operative AF)

Table 3

Occurrence of atrial fibrillation in the post-operative setting sorted by indication for transplant.

Indication	No post op A fib (n=147)	Post op A fib (n=27)	p value
Cystic fibrosis	19 (12.9%)	2 (7.4%)	0.42
COPD	25 (17.0%)	11 (40.7%)	0.005
Idiopathic Pulmonary Fibrosis (IPF)	46 (31.3%)	11 (40.7%)	0.34
Interstitial lung disease (except IPF)	39 (26.5%)	5 (18.5%)	0.38
Primary pulmonary hypertension	10 (6.8%)	0 (0%)	0.16
Lung Carcinoma	3 (2.0%)	0 (0%)	0.45
Alpha 1 Anti-trypsin deficiency	3 (2.0%)	1 (3.7%)	0.60
Congenital heart disease	2 (1.4%)	0 (0%)	0.54